HENRY A. ANDERSON M.D.

Occupational and Environmental Medicine
200 Lakewood Blvd., Madison, WI 53704 • (608) 241-1227

July 16, 2012

Robert McCoy
CASCINO VAUGHAN LAW OFFICES Ltd
220 SO ASHLAND AVE
CHICAGO IL 60607-5308

RE: Mr. Robert Kalis
Date of Birth: [redacted] 1937

Dear Mr. McCoy:

I am a Wisconsin licensed, board certified physician in occupational and environmental medicine and am currently the Chief Medical Officer for Occupational and Environmental Health and State Occupational and Environmental Epidemiologist with the Wisconsin Division of Public Health. I am a Diplomate of the American College of Preventive Medicine with a subspecialty in Occupational Medicine. I am also a Fellow in the American College of Epidemiology. I am a NIOSH certified "B" reader for interpreting chest x-rays using the ILO Pneumoconiosis Classification. For 39 years I have specifically studied asbestos exposure and the diseases it causes. Since 1972 I have conducted epidemiologic research on asbestos and human disease and published over 25 scientific journal articles addressing asbestos associated diseases. My background and experience is set forth in greater detail on my Curriculum Vitae. That and my list of prior testimony at deposition and trial have been provided previously. My fee is $200/hr for report preparation. I have spent one and one half hours reviewing materials and preparing this report.

The facts and data considered in formulating my report are:

1. Advocate Good Samaritan Hospital, Downers Grove, IL medical records and radiology reports;
2. "B" Reading of Henry A. Anderson, MD 9/19/1998 of a 6/22/1998 x-ray.
3. 1/30/1999 physician's report of Henry A. Anderson, MD.
4. "B" Reading of Steven Haber, MD 12/19/2011 of a 6/22/1998 x-ray.
5. Pulmonary function tests performed on 3/18/1999.
6. Work History and job sites summary provided by Cascino Vaughan Law Offices.

I evaluated a chest x-ray of 6/22/1998 using the ILO Pneumoconiosis Classification. That film was of quality 1 and showed parenchymal abnormalities consistent with pneumoconiosis. Small irregular opacities, size shape s/t in profusion 1/0, were present bilaterally in the lower lung zones. Circumscribed, "in profile" pleural plaque was present on the right chest wall, width "A" extent 1 and on left chest wall, width "A", extent 2. Calcified pleural plaques were present on both diaphragms. After reviewing an occupational history indicating work as an electrician, HVAC mechanic and stationary engineer with 12 years of asbestos exposure and his cigarette smoking

history, I issued a physicians report on 1/30/1999 concluding that Mr. Kalis had a condition consistent with bilateral asbestosis and bilateral asbestos-associated pleural disease.

A 12/19/2011 "B" reading by Dr. Haber interpreted a chest x-ray of 12/22/1998 as film quality 2 (scapulae overlap chest wall). He reported the presence of parenchymal abnormalities consistent with pneumoconiosis; consisting of irregular small opacities of shape/size t and t in profusion 1/1 bilaterally in the lower lung zones. "In profile" pleural plaque was present bilaterally width "b" on the right and width "b" on the left. "Face on" pleural plaque was seen bilaterally as well. Calcified pleural plaque was present on the left diaphragm. The overall extent of pleural plaque was extent 3 on the right and extent 3 on the left. He also recorded "OD" and noted focal scarring vs atelectasis in the right lower lobe.

Radiology and clinical assessment reports from Advocate Good Samaritan Hospital, Downers Grove, IL reported the presence of pleural thickening and mild chronic fibrotic interstitial changes.

Pulmonary function studies done showed abnormalities of obstructive lung disease and decreased DLCO. Mr. Kalis reported he smoked 2 to 2½ pack of cigarettes per day for 40 years, quitting in 1992.

I considered the Work History Summary and job site list provided by Cascino Vaughan Law Offices along with the medical records to identify any occupational substantial contributing causes of Mr. Kalis's disease.

Summary

My opinions concerning how the lungs function, how asbestos fibers enter the lungs and are distributed throughout the body and the abdomen and the prolonged period between exposure and disease progression that leads to the clinical recognition of asbestos diseases as well as the development of scientific knowledge about asbestos and disease discussed in this report are further detailed in affidavits of 2/27/2010, 3/28/2011 and 4/18/2011 as well as trial testimony I have provided in: Lemberger v General Motors Corporation, et al. Milwaukee county Case No.-CV-10416 7/17/08 – Volume 4 and 7/24/2008 – Volume 9; Gosz v American Standard, Inc., et al. Milwaukee County Case No 05-CV-9218, 11/7/2008; Winnemueller v. Foster Wheeler Energy Corporation, Milwaukee County Case No. 06-CV-000486 2/19/2009.

Based upon the facts and data considered, I have formed the following opinions concerning Mr. Kalis's medical condition. Within the bounds of medical certainty, it is my expert opinion that:

1. Mr. Kalis suffers from asbestosis/pulmonary fibrosis and asbestos associated pleural plaques with calcification;
2. His asbestosis/pulmonary fibrosis and pleural plaques with calcification were caused by his occupational exposure to asbestos;
3. All Mr. Kalis's asbestos exposure circumstances while employed as an electrician for 45 years, from 1957 to 2002 at industrial and commercial sites such as railroad companies and hospitals substantially contributed to cause his asbestosis and asbestos-associated pleural plaques. As an electrician, Mr. Kalis ran electrical conduit, pulled wire, and overhauled, repaired, and installed asbestos-containing motors and switch gear. Mr. Kalis worked cutting, stripping, and bending asbestos-insulated wire and cable using tools, including without limitation, needle-nosed pliers and drills. He also removed and drilled holes in asbestos insulation, wallboard, and tiles in order to access and run conduit. These

activities created asbestos debris, and generated and circulated airborne asbestos dust. Mr. Kalis also drilled through asbestos-insulated wallboard and fiberboard in order to pull wiring and install asbestos insulating films and papers on it. These activities generated airborne asbestos dust. Mr. Kalis installed asbestos refractory boilers. This work required him to attach the refractory by pounding it with a mallet, creating airborne asbestos dust. Mr. Kalis removed and replaced asbestos gaskets using tools, including without limitation, knives, scrapers, and electric-powered wire brushes. This work generated airborne asbestos dust. Mr. Kalis also dismantled, cleaned, and repaired asbestos-containing motors and other electrical equipment. He cleaned these motors using compressed air-hoses, which generated and circulated airborne asbestos dust. Mr. Kalis worked in close proximity to other tradesmen, including without limitation, insulators, pipefitters, and laborers working with asbestos-containing materials. Mr. Kalis worked in the immediate presence of insulators as they removed asbestos block insulation from boilers during repair work. This activity generated and circulated airborne asbestos dust. Mr. Kalis also worked under overhead cranes that possessed asbestos brakes. Engaging the breaks generated airborne asbestos dust.

4. Mr. Kalis is at substantial increased risk for malignant mesothelioma and lung cancer due to his occupational asbestos exposure.

My opinions stated in this report are held to a reasonable degree of scientific and medical certainty and are based upon: 1. My own 39 years of research and studies of asbestos diseases; 2. My professional training, experience and observations and; 3. Research and studies of other scientists, governmental bodies and professional organizations.

I declare under penalty of perjury that the foregoing is true and correct.

Very truly yours,

Henry A. Anderson, M.D.

Expert Materials of Dr. Henry Anderson for All CVLO MDL 875 Cases (updated May 3, 2012)

Cascino Vaughan Law Offices (CVLO) submits the following materials as part of the Rule 26 reports for all cases in which Dr. Henry Anderson submits a report.

1. Dr. Anderson's qualifications, including a list of all publications authored in the previous 10 years, is attached as Exhibit A.
2. A list of all cases in which, during the previous 4 years, Dr. Anderson has testified as an expert at trial or by deposition, is attached as Exhibit B.
3. The Affidavit of Henry Anderson, M.D. 2/27/2010, which includes testimony on the history of the knowledge of the dangers of asbestos, is attached as Exhibit C.
4. A statement of the compensation to be paid to Dr. Anderson for study and testimony in these cases, is attached as Exhibit D.

1/1/2001 - 3/6/2012

Henry A. Anderson, M.D.

TESTIMONY AT DEPOSITION

Jack Garverick - Goldberg Group 7 (3/29/2001); Marvin Boede et al. v WEPCO, et al. (4/20/01); Gerald Liebsch, Kordus v WR Grace, et al. (11/19/01); Marvin Boede et al (Krejcovic, Gerek). v WEPCO, et al.(1/8/02)**;** Laverne Hasse v Badger Mining et al. (3/14/02); State of Illinois, et al. vs. US Gypsum Co et al. (5/8/02); Paul P Coen vs Combustion Engineering, et al (8/20/02); Arno Knight v Owens-Illinois, Inc et al (9/4/02); Charles Barker v Dana Corp, et al. (9/27/02); Robinson v. Bartelt Insulation Company (2/12/03); Ernisse vs WEPCO et al. (3/11/03); Bowman vs Ford Motor Company (3/12/03); W.R.Grace/ZAI Science Trial (3/30/03); Halema vs WEPCO et al. (5/27/03).Glidden v. Georgia-Pacific Corporation (8/29/03);Gutmann v Wisconsin Electric Power Company, et al. (9/16/03); Vickman v Fort James Corp et al. (10/8/03); Clarence Abner, et al. v Frank Fulcon, et al.(1/9/04); Mr. Lloyd Poulson v. Georgia-Pacific Corp (1/30/04); Zaborski v Sprinkmann Sons Corporation, et al (3/18/04); Stoeger, et al. v. Bartelt Insulation Supply, Inc., et al.(4/8/04); DeJohn vs ACandS, Inc., et al.(5/12/04); William Griffin and Ben Dell Lee vs. Badger Mining Corporation, et al. (8/30/04); Hill, Brashear (Larry Scott) v. Bechtel Corp. et al. (9/15/04); Francis O'Flaherty, LaVerne C. Wieland v Badger Mining Corp. et al (2/1/05); Robert Krogh v Garlock (3/15/05); Hill v Becktel Corp, et al. (4/7/05); Pozarski v Certainteed Corporation et al. (5/5/05); Koke v WEPCO, et al (11/4/05); Cullen v. BSIS, et al (11/21/05); Roger Schultz v WEPCO, et al (2/16/06); Robert Cox et al v Minnesota Mining and Manufacturing Co. et. al. (William Staley) (3/28/06); Stacey Klewer Stanton v. Owens-Corning Fiberglas Corp., et. al. (4/21/06); Switalski v WEPCO, et al (5/18/06); Druke ,et al v American Standard, Inc. et al. (5/22/06); Antczak v Certainteed Corporation et al. (5/23/06); Kenny v. Commonwealth Edison, et al. (6/15/06); Richard Scheible v BSIS, et al. (8/17/06); Fittshur v Acoustech Supply Inc. et al. (10/2/06); Besaw/Greene v Bay Industries, et al.(10/4/06); Property Damage Asbestos Claimants Committee v WR Grace (3/30/07); Alyce Winnemueller v Employers Insurance Co of Wausau, et al (6/7/07); Audrey Streng v American Standard, Inc et al (8/2/07); Tatera v. FMC Corp., et al.(8/6/07); Loren Risse v Allied Insulation Supply Co., Inc et al. (8/16/07); Besaw/Green v Owens Illinois (9/11/07); Colleen Lemberger v Anchor Packing Co et al.(11/9/07); Roma Kurtz vs Allied Insulation Supply co Inc et al (12/18/07); Norman A. Johnson vs Sprinkmann Sons Corp., et al. and Jerome Kolinski v Fleming Materials, Inc., et al. (1/14/08); Francis Vandermeuse v Allied Insulation Supply Co., et al. (1/25/08); Richard Loos v Milwaukee Stove and Furnace, et al (5/21/08); Jean M. Dunk v Daimlerchrysler Corp., et al.(6/23/08); Mary Wagner v. BSIS Co et al.(10/8/08); Eske v Allied., et al. (10/14/08); Fredin v Bartelt Insulation Supply, et al (11/11/08); Ritchie v Foster Wheeler Corp, et al. (11/24/08); Kaminski v. American Standard, Inc, et al. (12/15/08); Kaminski v. American Standard, Inc, et al. (5/2/09); Tischer v American Standard, Inc, et.al. (7/9/09); Estate of Richard A. Jankoski v Marc Jay Bern and Napoli Bern Ripka, LLP et al (7/27/09); Carl Gnewuch v Allied Insulation Supply Co, Inc. et al (11/23/09) Bree v Allied Insulation Supply Co, Inc. et al. (12/17/09); Singer v. Sprinkmann Sons Corp, et al. (2/17/10); Plantiffs Ackerman T, Bathke JE, Vandermeuse FJ, Vandermause J, Ullman RA v. Allied Insulation Supply Corp et al.(4/7/10, 4/30/10); Provance vs Metropolitan Life Insurance Co. et al (5/3/10); Eurich v Allied Insulation Supply Inc et al (12/21/2010); Sorenson v Building Service Industrial Sales, Inc, et al. (12/22/2010); Josef Sercl v. Allied Insulation and Supply Co., Inc et al.(1/14/2011); June Calewarts v. Building Services Industrial Sales, Inc., et al.(3/31/2011); Dorothy Merline v. Building Services Industrial Sales, Inc., et al.(4/21/2011); Asbestos Products Liability Litigation MDL-875. (10/25/2011); Anderson v. Bechtel Corp et al, Dillman v Sprinkmann Sons Corp, Pleaugh,
v. A.W. Chesterton Company, et al.(12/15/2011); Loritz v. Building Service Industrial Sales, Inc., et al. (3/5/2012)**.** .

TESTIMONY AT TRIAL

Marvin Boede et al. v WEPCO, et al.(7/13/2001); Gerald Liebsch, Kordus v WR Grace, et al. (12/18/01); Laverne Hasse v Badger Mining et al. (4/16/02)**;** Bowman vs Ford Motor Company (3/15/03); James Schoonover vs Georgia-Pacific Corporation et al. (2/3/04); DeJohn vs ACandS, Inc., et al.(7/8/04); Arnold R. Norris v Sprinkmann Sons Corp., et al. (12/11/06). Lemberger v. Anchor Packing Company, et al.(7/17, 7/24, 08); Clarence Gosz v. American Standard, et al.(11/7/08); Lambert Winnemueller v Building Services Industrial Sales, et al (2/19/09); Eske v. Allied Insulation Supply Co., Inc. (4/23/10); Singer v. Sprinkmann Sons Corp, et al. (5/5/10); Streng v American Standard, et al. (1/13/2012).

STATE OF WISCONSIN)
) SS
COUNTY OF MILWAUKEE)

AFFIDAVIT OF HENRY A. ANDERSON, M.D.

I, Henry A. Anderson, M.D., being duly sworn on oath deposes and states as follows:

1. I am a board certified physician in occupational and environmental medicine and am currently the Chief Medical Officer for Occupational and Environmental Health and State Occupational and Environmental Epidemiologist with the Wisconsin Division of Public Health. *See* Curriculum Vitae attached hereto as Appendix 1.

2. I am also a Diplomate of the American College of Epidemiology.

3. I have been certified and recertified by NIOSH since 1985 as a "B" reader for interpreting chest x-rays for pneumoconiosis. I have specifically studied the risks of asbestos and its related diseases for over 35 years and over 25 of my 238 scientific publications address asbestos related diseases.

4. The opinions I express in this affidavit are stated within the bounds of reasonable medical probability.

5. I base my opinions upon the materials reviewed, my own research and studies of asbestos diseases, professional training, experience and observations, and on research and studies of other scientists and governmental bodies.

Asbestos Exposure and Disease

7. Asbestos has been recognized as a human health hazard causing injury to the body for nearly a century.

8. There are literally tens of thousands of medical articles written about all aspects of asbestos, its injurious properties, the results of which are expressed as different clinically diagnosable diseases including nonmalignant conditions such as pleural thickening, pleural plaques, and asbestosis, as well as malignant conditions such as lung cancer and mesothelioma.

9. The hazardous effects of asbestos are permanent, there are no curative treatments, and the clinical diseases caused by asbestos can gradually progress even after exposure ceases and can lead to disability and death.

10. Commercial production of asbestos insulation material began in 1874.

11. In the 1900's, inhalation of asbestos was first described in the medical literature as hazardous, causing permanent, progressive and irreversible lung tissue injury after inhalation severe enough to lead to death.

12. Inhalation of asbestos dust was recognized as hazardous leading to tissue damage well before the first case was published by Dr. Murray in 1906 and the tissue damage was given the name asbestosis.

13. In 1906, Auribault reported 50 deaths in France.

14. The second published case from Great Britain came in 1924. The tissue damage described in these reports was given the clinical disease name of asbestosis (scarring of the lungs). Reports of individual cases and case series such as these are

important to the practice of occupational medicine and preventive medicine because they alert clinicians and industries that existing industrial use of a material such as asbestos and dusty worksite conditions are hazardous.

15. Even before the full extent of the health hazard impact is known (how many of the exposed are ill, with what diseases and at what exposure levels) preventive, health protective actions such as dust suppression, consideration of material substitution, warnings, safety instructions to current workers and respiratory protection should be initiated by industry to reduce exposures and the subsequent risk of disease pending the outcome of comprehensive in-depth investigations the results of which may take time to develop.

16. The final confirmation of causal relationships for many occupational diseases develops as the result of many contributions over time and lags considerably behind the initial recognition of hazard because of the "latency period" between exposure and when disease is severe enough to be recognized as a clinical entity.

17. In the case of asbestos, confirmation of the case report observations came including from Dr. Merewether, the Chief Medical Inspector of Factories who conducted an inquiry and in 1930 published a detailed report "*The occurrence of pulmonary fibrosis and other pulmonary affections in asbestos workers,*" in the Journal of Industrial Hygiene. Multiple studies by the Factory Inspectorate and others had been undertaken and reported in 1930. In their 1930 study of 374 asbestos textile workers Drs. Merewether and Price found that among those with greater than 20 years exposure 80% had x-ray abnormalities.

18. The initial reports and regulatory efforts in Great Britain focused on asbestos

factory workers. However early on it was recognized that it was not specific occupations that caused the disease but rather any environment where asbestos fibers were released and inhaled could result in tissue damage.

19. In 1931, Drs Wood and Gloyne had accumulated 57 cases of asbestosis including a case in a 42 year old asbestos sawyer who had been exposed for 7 years from 1918-1928.

20. Three years later in 1934, Drs Wood and Gloyne had almost doubled the number of cases they had seen, accumulating 100 cases. They reported on them in the widely read general medical journal Lancet. Among the 33 male cases they reported cases without factory exposure including: an 18 year old van boy who for 2 ½ years during his spare time had mixed powered asbestos in an open yard; a middle-aged boiler riveter who had served his apprenticeship as a youth in a shop where asbestos was used for lagging pipes; and a man who had been employed handling asbestos mattresses in the open air at an aerodrome.

21. The first published case of asbestosis in the United States was in 1930 by Dr. Mills from Fondulac, Wisconsin and in the next few years other reports were published including cases from South Carolina reported by Dr. Lynch and Smith.

22. The Journal of the American Medical Association published highlights from the Merewether report and Dr. Mill's case in 1930.

23. In 1931 pleural fibrosis, plaques and calcification were the focus of a report in the journal Radiology by Dr. Sparks.

24. In 1932, the Wisconsin Industrial Commission sponsored a conference "*Effects*

of Dusts upon the Respiratory System" in Chicago and Dr. Russell, Surgeon of the United States Public Health Service and the US Bureau of Mines was a featured speaker. Dr. Russell reported a case of asbestosis in a man whose occupation for six years was cleaning and restoring the asbestos on pipes in a government hospital. He was compensated for his disability. Attendees of the conference included industry, workers' compensation insurance industry representatives and clinicians.

25. I have reviewed documents beginning in the 1930s from the archives of the Wisconsin Historical Society summarizing cases awarding compensation to asbestos victims.

26. By the early 1940s the scientific and medical literature documented that the lung tissue injury begins with the first exposure to asbestos fibers which lodge in the lungs and remain there but the triggered tissue response does not immediately result in clinical signs or symptoms, but gradually progresses until advanced sufficiently that clinical disease can be recognized.

27. The period of time between first exposure and when the disease process becomes clinically apparent is often described as the "latency period."

28. Prognosis, once disease occurred, was well described and because asbestosis was recognized as an important industrial disease it began appearing in standard medical textbooks.

29. The chapter on asbestosis in the Textbook *"Outlines of Industrial Medical Practice" by Dr Howard E. Collier,* published by Williams & Wilkins of Baltimore, MD in 1941 is one example and succinctly summarizes the understanding that:

a. asbestos risk is a dust risk;

b. Occupations at risk are numerous and fall into two categories, those associated with the manufacture of products and those with occasional or incidental use of asbestos in a dry state, in a considerable variety of industries;

c. Control of an isolated risk in industry is a matter to which industrial medical officers should give attention and applies particularly to work done in the store-rooms, to mixing and handling of dry asbestos…

d. When the dust has once entered the finer bronchioles, its removal is either impossible or, at best, a very slow process;

e. In the case of asbestosis the noxious substance remains almost indefinitely in the lung tissue and continues to produce its effects long after the inhalation has taken place;

f. Exposure to asbestos for less than five years can result in asbestosis sufficient to cause death;

g. Asbestosis definitely shortens life;

h. The fibrosis-producing period, that is to say, the period which elapses between the inhalation of the dust and the development of a serious degree of asbestosis is, as a rule, 5-11 years.

30. Since the initial reports of asbestosis, and consistent with the early recognition that it was asbestos fiber exposure and inhalation of dust that caused disease and not a specific occupation, disease has been documented occurring after exposure to asbestos in many different occupational settings, including mining of asbestos, manufacturing of asbestos products, asbestos pipe and boiler insulation application and removal in buildings as well as aboard ships, construction worker tradesmen such as carpenters and painters, plumbers and boiler repairmen, auto brake mechanics replacing and installing asbestos brake pads, janitorial and maintenance workers in buildings containing asbestos products, bystander tradesmen exposed while others worked directly with asbestos, family members living in the households of workers and those living near industrial facilities utilizing

asbestos.

Asbestos Exposure and the Disease Process

31. Asbestos related diseases are the product of multiple exposures to asbestos fibers inhaled through the lungs.

32. A small percentage of these fibers are retained in the lungs causing an insult to the tissue.

33. All exposures to asbestos fibers are considered to contribute to the disease process.

34. The disease process begins soon after the asbestos fibers are retained in the lung with early changes to biological cells that are not detectable to the exposed individual or their physician.

35. The "injury" or "damage" to human tissues begins after inhalation of asbestos fibers.

36. I use the term "injury" to describe both these.

37. It is my opinion to a reasonable degree of scientific and medical certainty that the injury begins soon after the inhalation of the fibers with changes to certain cells of the body which I describe as the "disease process."

38. The injury from asbestos is progressive and begins many years before the clinical diagnosis of different diseases caused by fibers can be made.

39. The asbestos injury may ultimately be expressed as different clinically diagnosable diseases including nonmalignant conditions such as pleural thickening, pleural plaques with and without calcification, and asbestosis, as well as malignant

conditions such as cancer or malignant mesothelioma.

40. Due to the gradual and progressive nature of the injury, signs and symptoms used in the diagnosis of an asbestos related disease manifest many years after the injury begins.

41. Consequently, diagnosis of an asbestos related disease occurs many years after the injury.

42. The disease process is not detectable by medical imaging or other usual diagnostic procedures until many years, usually more than ten, after the exposures have begun.

43. From my perspective as a physician in preventive medicine, it is not accurate to say there is no injury from asbestos until the disease is diagnosed.

44. Individual asbestos fibers are microscopic in size, and not detectable to the naked eye. Levels of airborne asbestos fibers capable of causing cancer are not necessarily visible.

45. In order to be visible, large amounts of airborne fibers must be present.

46. If one can see asbestos fibers in the air, a level far in excess of that capable of causing cancer is present.

47. As a result of its potency as a carcinogen and its difficulty to visualize, control of asbestos exposure is extremely difficult once asbestos has been liberated into the atmosphere.

48. Once airborne asbestos fibers are introduced into the workplace, exposure to the fiber is difficult to control.

49. Often invisible, the fiber contaminates floors and other surfaces.

50. Attempts to clean often unwittingly reintroduce fibers into the atmosphere where they can be inhaled and add to the lung fiber burden.

51. As fiber is transported in the air it can migrate undetected throughout a workplace.

52. These fibers can remain within a workplace for years posing a constant danger to anyone working within.

53. There is no known level of exposure to asbestos below which it can be said with scientific certainty that no risk of developing malignant mesothelioma exists.

54. Even brief or low level exposures to asbestos can result in mesothelioma.

Carcinogenicity of Asbestos Fibers

55. In addition to chronicling the increasing numbers of cases of asbestosis, the 1930s is usually identified as the time when the medical community identified other diseases disproportionately occurring among asbestos exposed individuals, especially cancer of the lung.

56. In 1933 Dr. Gloyne in a paper discussing the complications of asbestosis included a case of abdominal carcinoma and a squamous carcinoma of the pleura as complications. A year later (1934) Drs. Wood and Gloyne reported on 100 cases of asbestosis they had seen since 1928, including a case in a boiler-riveter, noting that of the 28 individuals who had died two had lung cancer and another had deposits of growth in the pleura.

57. These reports from Great Britain were followed in 1935 by a report from the

United States by Drs. Lynch and Smith from South Carolina that specifically discussed a possible correlation between asbestos exposure and lung cancer.

58. They detailed the case of an asbestos weaver who died from asbestosis at age 57 who also had lung cancer. That same year (1935) Dr. Gloyne focused a report on two cases of lung cancer occurring with asbestosis. Both cases were women, one age 35 and the other age 71. Both had worked in asbestos factories.

59. In 1936 Egbert and Geiger reported an additional case in the United States. This initial cluster of signaled the relationship between two rare diseases, asbestosis and lung cancer, and additional reports accumulated from other countries.

60. The Journal of the American Medical Association (JAMA) read by over 80% of physicians in the United States published an editorial in 1938 titled "Pneumoconiosis and Pulmonary Carcinoma."

61. Throughout the 1940s and 1950s deaths due to asbestosis mounted, regulations implemented and cases of lung cancer became more frequent.

62. As seen for asbestosis, the cases having both asbestosis and lung cancer included those with non-factory asbestos exposures.

63. In 1941, Drs. Holleb and Angrist reported two individuals whose duties always entailed the covering of steam and water pipes with asbestos fabric.

64. As identified in the earlier asbestosis reports the link between asbestos and lung cancer was reported in editorials in the Journal of the American Medical Association in 1938 as mentioned above but also in 1944 and 1949. It was also discussed in other widely read general medical journals.

65. In 1943 Wedler reported 4 cases of bronchial cancer and two others with malignant pleural growth out of 29 autopsies of asbestos workers. He concluded that lung cancer was the most common complication encountered in cases of asbestosis. The publication included a detailed review of the worldwide medical literature where he found 14 cases of malignant lung and pleural growths out of 92 autopsies for an incidence rate of around 16%. He reviewed each case and concluded that worker's compensation insurance coverage should be extended to include lung cancer in asbestos exposed workers.

66. In 1943, Germany made lung cancer related to asbestos compensable.

67. Dr. Merewether, The Chief Inspector of Factories in Great Britain produced annual reports on occupational disease being seen in Great Britain.

68. The report for 1947 was especially influential in solidifying the causal relationship between asbestos and lung cancer. In the 1947 annual report he found 31 lung cancers (13%) out of 231 men with asbestosis compared to only 91 (1.3%) lung cancers out of 6,884 men with silicosis.

69. The incidence rate he reported confirmed what Wedler reported in 1943. The general medical community was kept informed via additional editorials in JAMA in 1944 (*Environmental Cancer)* and 1949 (*Asbestosis and cancer of the lung.)*

70. In 1951, Stoll reported a New York case of a pipe coverer who had worked for the Works Progress Administration for 1 ½ years and for 4 ½ yeas as a pipe coverer in a shipyard. They concluded "*This association emphasizes the hazards of industrial exposure, the compensability of the cancerous process as well as the asbestosis, and the need of careful preventive measures.*"

71. Breslow in 1954 investigated the association between occupation, lung cancer and cigarette smoking in California among 518 lung cancer cases.

72. Seven occupational groups had significant excesses of lung cancer including the group of steam fitters-boilermakers, and asbestos workers which were ten times more likely to develop lung cancer than the comparison group. These excesses could not be explained by cigarette smoking.

73. The next year, 1955, Doll reported that 61 cases of lung cancer had been recorded in persons with asbestosis since the Lynch 1935 publication.

74. He conducted a cohort epidemiological study of asbestos factory workers and found 18 lung cancers among 105 deaths. He concluded *"From the data it can be concluded that lung cancer was a specific industrial hazard of certain asbestos workers and that the average risk among men employed for 20 or more years has been of the order of 10 times that experienced by the general population."* This study was published in a medical journal that is considered reliable and authoritative within the medical community and this article and its study were readily available in medical libraries throughout the United States.

75. In 1962 Cordova described lung cancer in asbestos exposed construction tradesmen such as a hood-carrier, iron worker, shipyard worker, bricklayer, carpenter and insulators.

76. In 1964 Dr. Selikoff from the Mount Sinai School of Medicine in New York City published a series of articles detailing the cancer risk among asbestos exposed pipe and boiler coverers. One was published in the Journal of the American Medical Association and another in the New England Journal of Medicine; the two most widely

read medical journals in the United States. These widely read and highly respected articles again set forth the links between asbestos exposure and non-malignant and malignant diseases, such as lung cancer and how smoking multiples the risk of lung cancer among asbestos exposed workers.

77. In 1964 an international conference was held at the New York Academy of Sciences titled "Biological Effects of Asbestos." In the 766 page proceedings published in 1965, nine papers dealt with asbestos and cancer and all supported the conclusion that asbestos caused lung cancer. Subsequently expert panels have issued consensus reports such as the 1997 Helsinki Criteria Consensus Report and the 1996 INSERM report confirming the causal link between asbestos and lung cancer, including mesothelioma. International organizations like the IARC, WHO and Collegium Ramazzini have reached the same conclusion and the WHO and Collegium called for the banning of the use of all asbestos.

Asbestos exposure, cigarette smoking and lung cancer

78. As detailed above, asbestos exposure causes lung cancer.

79. Tobacco smoke also causes lung cancer.

80. As documented by Dr. Selikoff, individuals who do not smoke but who are exposed to asbestos have 5 times the risk of contracting lung cancer as opposed to those who do not smoke and who have no asbestos exposure.

81. Individuals who smoke but who do not have asbestos exposure have 10 times the risk of getting lung cancer in comparison to those who neither smoke nor who have a history of asbestos exposure.

82. The studies show that those who have a history of asbestos exposure and smoke cigarettes have a greater than additive risk of developing lung cancer.

83. In Dr. Selikoff's studies the two risks fit a multiplicative model resulting in a 50 times greater risk of getting lung cancer among those who were exposed to asbestos and were cigarette smokers.

84. Dr. Hammond demonstrated in his studies that the risk of lung cancer due to cigarette smoking decreases each year after an individual ceases cigarette smoking.

85. In the Surgeon General's 1990 report on smoking cessation, after 10 years without smoking, the risk is substantially reduced approaching that of a non-smoker.

86. The risk of developing lung cancer remains steady after an individual ceases asbestos exposure.

87. In his 1968 report "*Asbestos exposure, Smoking and Neoplasia*" Dr. Selikoff reported that 49 (52%) of 94 deaths that occurred among 370 asbestos insulators were due to asbestos associated diseases (lung cancer, mesothelioma, asbestosis).

88. All forms of asbestos cause lung cancer in laboratory animals and humans.

89. OSHA and the US Environmental Protection Agency are responsible for regulating asbestos in the United States and have declared all forms of asbestos to be a known human carcinogen.

90. Internationally, the International Agency for the Research on Cancer (IARC) has also declared asbestos to be a known human carcinogen. Many countries have banned the use of asbestos because of the hazard it presents.

91. While the early necropsy reports of asbestosis deaths found frequent

concomitant lung cancer suggesting only those asbestos exposed workers with asbestosis were at risk for the development of lung cancer, subsequent research studies and international panels of experts such as the Helsinki Criteria Consensus Report and the INSERM joint expert analysis group have confirmed that it is cumulative exposure to asbestos not the presence of asbestosis that causes lung cancer and that asbestos can cause lung cancer in the absence of asbestosis.

Asbestos exposure and mesothelioma

92. Beginning in 1943 attention was drawn to reports of an unusual type of lung cancer, malignant pleural mesothelioma being associated with asbestos exposure.

93. In 1952, Smith published a paper in the Archives of the Industrial Hygiene and Medicine which reviewed studies conducted in Britain and elsewhere in Europe, including discussion of asbestos and mesothelioma.

94. By 1960, epidemiologic studies confirmed the strong association between asbestos exposure and malignant mesothelioma. Of particular note was that disease occurred not only among workers handling asbestos but among those only exposed environmentally or as bystanders in the vicinity of workers.

95. The only known occupational or environmental cause of malignant pleural mesothelioma in the United States is the exposure to, and inhalation of asbestos fibers.

96. From the 1950s on, more and more studies were reported and published that confirmed again and again these prior study results linking asbestos exposure with the development of asbestosis, pleural plaques, lung cancer and mesothelioma. These included Dr. Irving Selikoff's articles on New York and New Jersey asbestos insulators published in

the Journal of the American Medical Association and the New England Journal of Medicine from the mid 1960's on.

97. In 1964 an international conference "*Biological Effects of Asbestos*" was hosted by the New York Academy of Sciences. Chaired by Dr. Selikoff, research experts from around the world attended and presented the most recent findings. The conference proceeding filled 765 pages in a special issue of the Annals of the New York Academy of Sciences and remains a major historical reference on asbestos disease. These widely read and highly respected articles again set forth the causal link between asbestos exposure and non-malignant and malignant diseases, and how cigarette smoking multiplies these risks.

98. The studies by Dr. Selikoff and others that found the interaction between asbestos and cigarette smoking in the causation of lung cancer did not find any relationship between cigarette smoking and the risk of malignant mesothelioma.

99. Beginning in the 1930s to 1940s, occupational health and the general medical literature documented the hazard of asbestos inhalation, and that the injury caused by asbestos could be expressed as several clinical diseases including asbestosis, lung cancer and mesothelioma.

100. The scientific articles in Appendix 2 are examples of the information that was available regarding the link between asbestos exposure and disease. This information was published in the frequently read general medical journals such as the Journal of the American Medical Association, the American Journal of Public Health and the New England Journal of Medicine.

Occupational Safety and Health Regulation in Wisconsin

101. Since at least 1913, the Wisconsin Industrial Commission (Commission) has been publishing a list of toxic substances which require worker protection.

102. Each material regulated had "a characteristically harmful effect on some part, or parts, of the human body."

103. The provisions of the code applied to "all places of employment and public buildings" as defined by the code to include such places as those at which business is carried on or at which any person is directly or indirectly employed by another.

104. In 1928, increasing mortality and disability rates were revealed by the record of cases settled under the Workmen's Compensation Act.

105. The increases indicated the hazard to the health of workmen was increasing. In response, the Commission formed an advisory committee to review the existing general orders and recommend changes.

106. In 1932 that extensive review resulted in the issuance of expanded "General Orders on dusts, fumes, vapors and gases".

107. In 1947, the Industrial Commission of Wisconsin modified its General Order 2002 with regard to Dusts, Fumes, Vapors and Gases to include asbestos as a toxic substance. The order established harmful exposure levels for asbestos at 5 million particles per cubic foot, stating that "concentrations that equal or exceed [that level] shall constitute harmful exposures or harmful concentrations."

108. Asbestos continued to be listed as a toxic substance in subsequent versions of

the order, including the 1955 revision.

109. The advisory committee to the commission noted that the nature and effect of injuries caused by inhalation hazards was "the subject of much study and investigation by the medical profession and others" and that data had been "widely published in treatises, in medical journals, and in government and professional bulletins."

110. Many of the historic records of the Wisconsin Industrial Commission meetings, sponsored conferences and correspondence are maintained by the Wisconsin Historical Society in their library archives. These demonstrate that the Commission was active in promoting worker safety and health and kept informed of advancements concerning asbestos health risks.

111. The asbestos Threshold Limit Value (TLV) or Maximum Contaminant Level (MCL) which were determined by the American Conference of governmental Industrial Hygienists (ACGIH) were not intended to be protective against the development of cancer associated with asbestos. Standards such as those adopted by Wisconsin were based on the ACGIH values designed to reduce the incidence of asbestosis but were not intended or sufficient to prevent cancer. This was detailed by Dr. Stokinger, who chaired the ACGIH TLV committee, in 1956 in the journal of the American Industrial Hygiene Association. He stated, *"There is still one group of substances for which some method should be devised for establishing safe air standards – the industrial cancerigens. Thus far the question has been sidestepped completely. As a result, with one exception, nickel carbonyl, limits taking into consideration potential cancerigenicity have not been assigned."* He goes on to propose a method to address carcinogenicity by reducing the TLV by a factor of 100 to 500.

112. At the 1965 New York Academy of Sciences international meeting of asbestos experts it was recognized by the asbestos industry that the standards at the time were not "safe."

113. Dr. Addingley of British Belting and Asbestos Ltd stated that "*We (British asbestos industry) do not believe there is any safe limit.*" Speaking about the American limit of 5 million particles per cubic foot being a "safe" standard he said, "*We know that there is no scientific basis for that limit whatever.*"

114. After the 1970 passage of the federal Occupational Safety and Health Act and the subsequent establishment of the US Occupational Safety and Health Administration (OSHA), OSHA developed national standards and enforcement programs. With the onset of federal activity, the primary thrust of occupational safety and health in Wisconsin shifted from State to Federal implementation.

115. Although many states had occupational safety and health standards regulating asbestos, before the Occupational Safety and Health Act (OSHA) of 1970, which became effective on April 28, 1971, OSHA provided the United States with a mechanism to develop national standards. One of the first national standards addressed by OSHA was that for asbestos.

116. On May 29, 1971 OSHA adopted a standard for asbestos and on December 7, 1971 lowered that standard through an emergency standard.

117. On January 12, 1972 OSHA issued another emergency standard covering the ship repairing, shipbuilding, ship breaking and longshoring industries. Since 1972 the standard has been lowered repeatedly and the current OSHA standard of 0.1 fibers per

cubic centimeter was promulgated in 1994.

118. In the Federal Register (Vol. 59:153 August 10, 1994 p 40967) announcing the final standard OSHA recognized; *"The 0.1 f/cc level leaves a remaining significant risk (calculated to be 3.4 excess cancers per 1000 workers), but that OSHA believes this (the standard) is the practical lower limit of feasibility for measuring asbestos levels reliably."* Because of the remaining cancer risk described by OSHA, it has been proposed that asbestos use be banned and many countries have already done so.

119. The World Health Organization supports a ban on all uses of asbestos.

120. A cubic centimeter is about the size of a sugar cube. Respirable asbestos fibers are so small that billions of fibers could fit into the space of a single sugar cube.

Summary

121. Any exposure to asbestos adds to the risk of developing asbestos disease.

122. There is no known level of exposure to asbestos below which it can be said with scientific certainty that no risk of developing cancer exists.

123. Asbestos is one of only a few agents classified as a "known human carcinogen."

124. In my opinion, with reasonable medical certainty, by the decades of the 1930s and 1940s it was known or could have been known by the medical community, industry management, workers' compensation insurers and occupational regulatory authorities that asbestos dust inhalation posed a significant human health hazard and that asbestos fibers penetrated deep into the lungs where they persisted, could not be removed, led to irreversible and progressive tissue damage which caused diseases which were incurable

and could lead to disability and death.

125. In the 1930s and 1940s, preventive medicine principles and practices were known and intervention actions available which could reduce the asbestos disease risks.

126. My opinions concerning how the lungs function, how asbestos fibers enter the lungs and the prolonged period between exposure and disease progression that leads to the clinical recognition of asbestos diseases as well as the development of scientific knowledge about asbestos and disease discussed in this report are further detailed in trial testimony I have provided in:

a. *Lemberger v. General Motors Corporation, et al.*, Milwaukee County Case No.-CV-10416, 7/17/08 - Volume 4 and 7/24/2008 - Volume 9;
b. *Gosz v. American Standard, Inc., et al.*, Milwaukee County Case No 05-CV-9218, 11/7/2008;
c. *Winnemueller v. Foster Wheeler Energy Corporation*, Milwaukee County Case No. 06-CV-000486, 2/19/2009.

127. The risk of asbestos exposure is not limited to workers in occupations who use or remove asbestos containing materials. Bystanders and persons working in facilities contaminated by asbestos are also at risk for developing asbestos related diseases. The risk of asbestos disease from contaminated non occupational settings, such as household laundry or community settings near facilities using large quantities of asbestos is also documented.

128. Industrial hygiene dust exposure measurements have been made by numerous investigators and document the occurrence of significant, intermittent, peak exposures to asbestos during asbestos-containing material application, repair and removal. The resulting disbursement and inhalation of these fibers is the public health risk which

129. All US regulatory agencies recognize the asbestos exposure hazard potential presented by work utilizing or disturbing asbestos containing products.

130. Within the bounds of medical certainty, it is my expert opinion that:

a. All exposures to asbestos substantially contribute to the development of carcinoma of the lung, asbestosis, calcified pleural plaques, and malignant mesothelioma; and

b. All asbestos types, chrysotile, amosite, crocidolite, tremolite are capable of, and have caused lung cancers, asbestosis, and mesothelioma in humans.

111. My opinions stated above are based upon:

a. My own 35 years of research and studies of asbestos diseases;

b. My professional training, experience and observations and;

c. Research and studies of other scientists, governmental bodies and professional organizations.

Further, Affiant sayeth naught.

Henry A. Anderson, M.D.

Subscribed and sworn to and before me
This 27 day of Feb, 2010.

Notary Public
Com Exp 12-02-12

KAREN M THOMPSON NOTARY PUBLIC STATE OF WISCONSIN

Appendix 2

Anderson Reliance Materials

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295. Dr. Anderson reserves the right to add additional publications to this list as needed.

Henry A. Anderson, M.D.

2012 Compensation Schedule

Preparation of expert reports	$200	per hour
Deposition testimony	$400	per hour (**evening**s)
	$500	per hour (work hours)
Out-of-state consultation	$2500	per day
Appearance at trial	greater	of $400 per hour or
	$2500	per day